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08/444,791	05/19/1995	MANFRED BROCKHAUS	A947-US-DIV4/01017/40451C	5613
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AMGEN INC. LAW DEPARTMENT 1201 AMGEN COURT WEST SEATTLE, WA 98119			EXAMINER SCHWADRON, RONALD B	
			ART UNIT	PAPER NUMBER
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**Please find below and/or attached an Office communication concerning this application or proceeding.**

The time period for reply, if any, is set in the attached communication.

# Office Action Summary

**Application No.**

08/444,791

**Applicant(s)**

BROCKHAUS ET AL.

**Examiner**

Ron Schwadron, Ph.D.

**Art Unit**

1644

**-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --**  
**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) ☐ Responsive to communication(s) filed on \_\_\_\_.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) ☒ Claim(s) 125, 127-130, 132, 148, 149, 155-159 and 213-273 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 125, 127-130, 132, 148, 149, 155-159, 213-273 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

**Priority under 35 U.S.C. § 119**

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
- ☐ Certified copies of the priority documents have been received.
  - ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_.
  - ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

**Attachment(s)**

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftperson's Patent Drawing Review (PTO-948)
- 3) ☒ Information Disclosure Statement(s) (PTO/SF/08)  
Paper No(s)/Mail Date 12/1/08 and 9/13/07
- 4) ☐ Interview Summary (PTO-413)  
Paper No(s)/Mail Date: \_\_\_\_
- 5) ☐ Notice of Informal Patent Application
- 6) ☐ Other: \_\_\_\_

1. Applicant's election with traverse of the species pCD4Hy1 in the reply filed on 12/18/09 is acknowledged. The traversal is on the ground(s) that are stated. This is not found persuasive because the searching of additional species would place an undue burden on the Examiner.

The requirement is still deemed proper and is therefore made FINAL.

2. The amendment filed 8/30/07 is objected to under 35 U.S.C. 132(a) because it introduces new matter into the disclosure. 35 U.S.C. 132(a) states that no amendment shall introduce new matter into the disclosure of the invention. The added material which is not supported by the original disclosure is as follows.

The addition of the term "incorporated by reference" does not find support in the specification as originally filed.

The addition of reference to the deposited vector PTA 7942 has no support in the specification as originally filed. Regarding applicants comments and the Lesslauer declaration, the entire sequence of the deposited sequence needs to be disclosed and applicant should point out where said entire sequence was described in the specification as originally filed. The fact that said construct was made is irrelevant if said construct is not disclosed in the specification as originally filed. There is currently no disclosure in the specification of said construct in the specification as originally filed.

Applicant is required to cancel the new matter in the reply to this Office Action.

3. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

4. The rejection of claims 204-207,209-211 under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention for the reasons

elaborated in the previous Office Action is withdrawn in view of the cancellation of said claims.

5. Claims 125,127-130,132,148,149,155-159,213-223,233-252,263-265,268-271 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

There is no support in the specification as originally filed for the recitation of section (b) in claims 125 or sections (b) and (c) in claim 233. The specification (page 35) discloses use of a specific unidentified HL60 cDNA library to isolate cDNA encoding the TNFR 75kD. However, the claims encompass use of HL60 cDNA libraries from any HL60 cell line. However, cDNA libraries made from different HL60 cell lines will differ in DNA content due to spontaneous mutation found in HL60 cells (for example, see Monnat, abstract). Thus, the specification discloses a specific library with specific sequences wherein the claims encompass use of HL60 libraries that contain different sequences. In addition, the specification discloses that the search of said cDNA library yielded the CDNA clone of Figure 4. The claims encompass sequences other than that disclosed in Figure 4.

There is no support in the specification as originally filed for claims that recite additional subsequences in the sequence of claim 125/233, as per above, the specification discloses that the search of said HL60 cDNA library yielded the CDNA clone of Figure 4. The claims encompass sequences other than that disclosed in Figure 4.

There is no support in the specification as originally filed for claim 129 because claim 125 has been amended to recite the sequence recited in claim 129 and the claim now encompasses molecules with two copies of said sequence which are not disclosed in the specification as originally filed.

There is no support in the specification as originally filed for recitation in claims 214/243 of the plasmid PTA 7942 for essentially the same reasons as stated in paragraph 2 of this Office action.

6. Claims 125,127-130,132,148,149,155-159,213,233-242,262,263,268,269 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The specification does not provide adequate written description of the claimed invention. The legal standard for sufficiency of a patent's (or a specification's) written description is whether that description "reasonably conveys to the artisan that the inventor had possession at that time of the . . . claimed subject matter", *Vas-Cath, Inc. v. Mahurkar*, 19 U.S.P.Q.2d 1111 (Fed. Cir. 1991). In the instant case, the specification does not convey to the artisan that the applicant had possession at the time of invention of the claimed inventions.

The only nucleic acid encoding a sequence comprising soluble portions of insoluble TNF binding proteins of a TNF 75 kD receptor disclosed in the specification are those disclosed in the Figures. The claims recite that the nucleic acid is the same as that found in an unspecified HL-60 cell line. However, cDNA libraries made from different HL60 cell lines will differ in DNA content due to spontaneous mutation found in HL60 cells (for example, see Monnat, abstract). Thus, the claims would encompass unknown and undescribed mutants and variants of the specific sequences disclosed in the specification.

Thus, the written description provided in the specification is not commensurate with the scope of the claimed inventions. In view of the aforementioned problems regarding description of the claimed invention, the specification does not provide an adequate written description of the invention claimed herein. See *The Regents of the University of California v. Eli Lilly and Company*, 43 USPQ2d 1398, 1404-7 (Fed. Cir. 1997). In *University of California v. Eli Lilly and Co.*, 39 U.S.P.Q.2d 1225 (Fed. Cir. 1995) the inventors claimed a genus of DNA species encoding insulin in different vertebrates or mammals, but had only described a single species of cDNA which encoded rat insulin. The court held that only the nucleic acids species described in the specification (i.e. nucleic acids encoding rat insulin) met the description requirement and

that the inventors were not entitled to a claim encompassing a genus of nucleic acids encoding insulin from other vertebrates, mammals or humans, *id.* at 1240. In the instant case, the specification has disclosed a single nucleic acid encoding a 55kD TNF receptor with the nucleic acid sequence disclosed in Figure 1. The Federal Circuit has held that if an inventor is "unable to envision the detailed constitution of a gene so as to distinguish it from other materials. . .conception has not been achieved until reduction to practice has occurred", *Amgen, Inc. v. Chugai Pharmaceutical Co, Ltd.*, 18 U.S.P.Q.2d 1016 (Fed. Cir. 1991). Attention is also directed to the decision of *The Regents of the University of California v. Eli Lilly and Company* (CAFC, July 1997) wherein is stated: The description requirement of the patent statute requires a description of an invention, not an indication of a result that one might achieve if one made that invention. See *In re Wilder*, 736 F.2d 1516, 222 USPQ 369, 372-373 (Fed. Cir. 1984) (affirming rejection because the specification does "little more than outlin[e] goals appellants hope the claimed invention achieves and the problems the invention will hopefully ameliorate."). Accordingly, naming a type of material generally known to exist, in the absence of knowledge as to what that material consists of, is not a description of that material.

Thus, as we have previously held, a cDNA is not defined or described by the mere name "cDNA," even if accompanied by the name of the protein that it encodes, but requires a kind of specificity usually achieved by means of the recitation of the sequence of nucleotides that make up the cDNA. See *Fiers*, 984 F.2d at 1171, 25 USPQ2d at 1606.

7. Regarding priority for the claimed inventions and the application of prior art, the claimed nucleic acids encoding fusion proteins are not disclosed in the Swiss priority documents. SEQ ID. NO: 27 is not disclosed in said applications. The vectors recited in the claims are also not disclosed in said applications. Also, the correct sequence for the DNA encoding the TNF 75 kD receptor (as per page 35, last paragraph of the specification) is not disclosed in said applications.

8. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and

the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

9. The rejection of claims 204-207,209-211 under 35 U.S.C. 103(a) as being unpatentable over Schall et al. in view of Capon et al. (US Patent 5428130) for the reasons elaborated in the previous Office action is withdrawn in view of the cancellation of said claims.

10. Claims 224-232,253-261,266,267,272,273 are rejected under 35 U.S.C. 103(a) as being unpatentable over Smith et al. (Science, 1990) in view of Capon et al. (US Patent 5428130).

Smith et al. teach DNA encoding an insoluble (eg. membrane bound) 75 kD TNF receptor that has the amino acid sequence of SEQ. ID. NO:27 (see page 1021). Smith et al. teach the extracellular portion of said molecule (see abstract). The extracellular portion of the membrane bound molecule would be a soluble portion of said molecule. Smith et al. do not teach a nucleic acid encoding an Ig/soluble portion of a 75kD TNF receptor. Capon et al. teach DNA encoding Ig/ligand binding fusion proteins (see column 5). Capon et al. teach that the Ig/ligand binding fusion protein can contain the soluble portion of a cell surface receptor (eg. the receptor minus the transmembrane and cytoplasmic domains, see column 8, first complete paragraph). Capon et al. teach that the DNA encoding the Ig portion of the fusion protein can contain at least the hinge, CH2 and CH3 domains of the constant region of an Ig heavy chain or the Fc portion of the heavy chain (see column 10, second paragraph). Capon et al. teach vectors /host cells containing said DNA and use of said cells to produce fusion protein (see column 26-30). Capon teach the use of IgG-1 constant region in said fusion proteins (see claim 3). Capon et al. teach that the ligand binding portion of the Ig/ligand binding fusion protein can be derived from a wide variety of different known cell surface receptors (see column 7, third paragraph from bottom). It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have created the claimed invention because Smith et al. teach the nucleic acid sequence encoding an insoluble (eg. membrane bound) 75kD TNF receptor while Capon et al. teach DNA encoding soluble Ig/ligand binding fusion proteins wherein the ligand binding protein is a

soluble portion derived from a cell surface receptor. One of ordinary skill in the art would have been motivated to do the aforementioned because Capon et al. teach that the ligand binding portion of the Ig/ligand binding fusion protein can be derived from a wide variety of different known cell surface receptors (see column 7, third paragraph from bottom) and that said fusion proteins have a variety of uses (see column 4). The IgG1 constant region fragment encoding nucleic acids of pCD4Hgamma1 appear to be the art known nucleic acids encoding the portion of the human IgG1 constant region as per disclosed in Capon et al.

Regarding applicants comments about unexpected results, the claimed inventions are drawn to nucleic acids, not proteins. There is no evidence of record regarding unexpected results and the claimed invention (aka nucleic acids).

11. Claims 224-232,253-261,266,267,272,273 are rejected under 35 U.S.C. 103(a) as being unpatentable over Smith et al. (US Patent 5,395,760) in view of Capon et al. (US Patent 5428130).

Smith et al. teach DNA encoding an insoluble (eg. membrane bound) 75 kD TNF receptor that has the amino acid sequence of SEQ. ID. NO:27 (see Figure 2). Smith et al. teach the soluble extracellular portion of said molecule (see column 4). Smith et al. teach DNA encoding an Ig fusion molecule. Smith et al. do not teach a nucleic acid encoding an Ig/soluble portion of a 75kD TNF receptor wherein the IG portion lacks the first domain of the constant region. Capon et al. teach DNA encoding Ig/ligand binding fusion proteins (see column 5). Capon et al. teach that the Ig/ligand binding fusion protein can contain the soluble portion of a cell surface receptor (eg. the receptor minus the transmembrane and cytoplasmic domains, see column 8, first complete paragraph). Capon et al. teach that the DNA encoding the Ig portion of the fusion protein can contain at least the hinge, CH2 and CH3 domains of the constant region of an Ig heavy chain or the Fc portion of the heavy chain (see column 10, second paragraph). Capon et al. teach vectors /host cells containing said DNA and use of said cells to produce fusion protein (see column 26-30). Capon teach the use of IgG-1 constant region in said fusion proteins (see claim 3). Capon et al. teach that the ligand binding portion of the Ig/ligand binding fusion protein can be derived from a wide variety of different known cell surface receptors (see column 7, third paragraph from bottom). It would have been



prima facie obvious to one of ordinary skill in the art at the time the invention was made to have created the claimed invention because Smith et al. teach the nucleic acid sequence encoding an insoluble (eg. membrane bound) 75kD TNF receptor and DNA encoding Ig fusion proteins containing said molecule while Capon et al. teach DNA encoding soluble Ig/ligand binding fusion proteins wherein the DNA encoding the Ig portion of the fusion protein can contain at least the hinge, CH2 and CH3 domains of the constant region of an Ig heavy chain and wherein the ligand binding protein is a soluble portion derived from a cell surface receptor. One of ordinary skill in the art would have been motivated to do the aforementioned because Capon et al. teach that the ligand binding portion of the Ig/ligand binding fusion protein can be derived from a wide variety of different known cell surface receptors (see column 7, third paragraph from bottom) and that said fusion proteins have a variety of a uses (see column 4). The IgG1 constant region fragment encoding nucleic acids of pCD4Hgamma1 appear to be the art known nucleic acids encoding the portion of the human IgG1 constant region as per disclosed in Capon et al.

Regarding applicants comments about unexpected results, the claimed inventions are drawn to nucleic acids, not proteins. There is no evidence of record regarding unexpected results and the claimed invention (aka nucleic acids).

12. Claims 125,127-130,132,148,149,155-159,213-223,233-252,262-265,268-271 are rejected under 35 U.S.C. 103(a) as being unpatentable over Dembic et al. (Cytokine, 1990) in view of Capon et al. (US Patent 5428130).

Dembic et al. teach DNA encoding an insoluble (eg. membrane bound) 75 kD TNF receptor that is derived from HL60 cells and that encodes the various peptide fragments recited in the claims (see page 231, second column). Dembic et al. teach the extracellular portion of said molecule (see abstract). The extracellular portion of the membrane bound molecule would be a soluble portion of said molecule. Dembic et al. do not teach a nucleic acid encoding an Ig/soluble portion of a 75kD TNF receptor. Whilst the identity of the sequence encoding the TNF receptor 75 kD in the construct of claim 214 is unclear, since it was apparently derived from HL60 cells it will be considered as encoding the same HL60 sequence as per disclosed in Dembic et al. Capon et al. teach DNA encoding Ig/ligand binding fusion proteins (see column 5).

Capon et al. teach that the Ig/ligand binding fusion protein can contain the soluble portion of a cell surface receptor (eg. the receptor minus the transmembrane and cytoplasmic domains, see column 8, first complete paragraph). Capon et al. teach that the DNA encoding the Ig portion of the fusion protein can contain at least the hinge, CH2 and CH3 domains of the constant region of an Ig heavy chain or the Fc portion of the heavy chain (see column 10, second paragraph). Capon et al. teach vectors /host cells containing said DNA and use of said cells to produce fusion protein (see column 26-30). Capon teach the use of IgG-1 constant region in said fusion proteins (see claim 3). Capon et al. teach that the ligand binding portion of the Ig/ligand binding fusion protein can be derived from a wide variety of different known cell surface receptors (see column 7, third paragraph from bottom). It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have created the claimed invention because Dembic et al. teach the nucleic acid sequence encoding an insoluble (eg. membrane bound) 75kD TNF receptor while Capon et al. teach DNA encoding soluble Ig/ligand binding fusion proteins wherein the ligand binding protein is a soluble portion derived from a cell surface receptor. One of ordinary skill in the art would have been motivated to do the aforementioned because Capon et al. teach that the ligand binding portion of the Ig/ligand binding fusion protein can be derived from a wide variety of different known cell surface receptors (see column 7, third paragraph from bottom) and that said fusion proteins have a variety of a uses (see column 4). The IgG1 constant region fragment encoding nucleic acids of pCD4Hgamma1 appear to be the art known nucleic acids encoding the portion of the human IgG1 constant region as per disclosed in Capon et al.

Regarding applicants comments about unexpected results, the claimed inventions are drawn to nucleic acids, not proteins. There is no evidence of record regarding unexpected results and the claimed invention (aka nucleic acids).

13. Claims 125,127-130,132,148,149,155-159,213-223,233-252,262-265,268-271 are rejected under 35 U.S.C. 103(a) as being unpatentable over Smith et al. (US Patent 5,395,760) in view of Hohmann et al. (J. Biol. Chem., 1989) and Capon et al. (US Patent 5428130).

Smith et al. teach DNA encoding an insoluble (eg. membrane bound) 75 kD TNF receptor that has the amino acid sequence of SEQ. ID. NO:27 (see Figure 2). Smith et al. teach the soluble extracellular portion of said molecule (see column 4). Smith et al. teach DNA encoding an Ig fusion molecule. Smith et al. do not teach a nucleic acid encoding an Ig/soluble portion of a 75kD TNF receptor wherein the Ig portion lacks the first domain of the constant region and wherein the DNA encodes the 75 kD TNF receptor in HL-60 cells. Smith et al. teach that nucleic acids encoding the 75 kD TNF receptor can be isolated from mammalian cells that express said receptor and method for isolating said DNA (see columns 5-6). Hohmann et al. teach that HL-60 cells express the TNF 75 kD receptor (see page 14929). Whilst the identity of the sequence encoding the TNF receptor 75 kD in the construct of claim 214 is unclear, since it was apparently derived from HL60 cells it will be considered as encoding the same HL60 sequence as found in said cells. Capon et al. teach DNA encoding Ig/ligand binding fusion proteins (see column 5). Capon et al. teach that the Ig/ligand binding fusion protein can contain the soluble portion of a cell surface receptor (eg. the receptor minus the transmembrane and cytoplasmic domains, see column 8, first complete paragraph). Capon et al. teach that the DNA encoding the Ig portion of the fusion protein can contain at least the hinge, CH2 and CH3 domains of the constant region of an Ig heavy chain or the Fc portion of the heavy chain (see column 10, second paragraph). Capon et al. teach vectors /host cells containing said DNA and use of said cells to produce fusion protein (see column 26-30). Capon teaches the use of IgG-1 constant region in said fusion proteins (see claim 3). Capon et al. teach that the ligand binding portion of the Ig/ligand binding fusion protein can be derived from a wide variety of different known cell surface receptors (see column 7, third paragraph from bottom). It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have created the claimed invention because Smith et al. teach the nucleic acid sequence encoding an insoluble (eg. membrane bound) 75kD TNF receptor and DNA encoding Ig fusion proteins containing said molecule and Smith et al. teach that nucleic acids encoding the 75 kD TNF receptor can be isolated from mammalian cells that express said receptor and method for isolating said DNA whilst Hohmann et al. teach that HL-60 cells express the TNF 75 kD receptor while Capon et al. teach DNA encoding soluble Ig/ligand binding fusion proteins wherein the DNA encoding the Ig portion of the fusion protein can

contain at least the hinge, CH2 and CH3 domains of the constant region of an Ig heavy chain and wherein the ligand binding protein is a soluble portion derived from a cell surface receptor. One of ordinary skill in the art would have been motivated to do the aforementioned because Capon et al. teach that the ligand binding portion of the Ig/ligand binding fusion protein can be derived from a wide variety of different known cell surface receptors (see column 7, third paragraph from bottom) and that said fusion proteins have a variety of uses (see column 4) and Smith et al. teach that nucleic acids encoding the 75 kD TNF receptor can be isolated from mammalian cells that express said receptor and method for isolating whilst Hohmann et al. teach that HL-60 cells express the TNF 75 kD receptor. The IgG1 constant region fragment encoding nucleic acids of pCD4Hgamma1 appear to be the art known nucleic acids encoding the portion of the human IgG1 constant region as per disclosed in Capon et al.

Regarding applicants comments about unexpected results, the claimed inventions are drawn to nucleic acids, not proteins. There is no evidence of record regarding unexpected results and the claimed invention (aka nucleic acids).

14. No claim is allowed.

15. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Ron Schwadron, Ph.D. whose telephone number is (571)272-0851. The examiner can normally be reached on Monday-Thursday 7:30-6:00 pm. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on 571 272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300. Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO

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/Ron Schwadron/  
Ron Schwadron, Ph.D.  
Primary Examiner  
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